Perioperative Ischemic Complications of the Brain After Carotid Endarterectomy

Hebb MO, Heiserman JE, Forbes KPN, et al. Neurosurgery 2010;67:286-94.

Conclusion: Carotid endarterectomy (CEA) is associated with a low rate of ischemic complications both clinically and with magnetic resonance (MR) diffusion-weighted imaging (DWI).

Summary: CEA is perhaps the most well studied procedure in vascular surgery. With carotid artery stenting, the incidence of "cerebral infarction" that is asymptomatic but detected by MR DWI can be high, and can exceed 50% even when cerebral protection devices are used. The authors present a small series of CEAs in whom the patients were evaluated postoperatively, both clinically and with routine MR DWI. Indications for surgery included symptomatic stenosis >50% and asymptomatic stenosis >60%. Two-thirds of the patients in this series were asymptomatic. Operations were performed by two neurosurgeons using what most vascular surgeons would consider relatively ponderous techniques for the performance of CEA, using an operative microscope for dissection of the carotid artery and performance of the endarterectomy and closure of the artery. Shunts were rarely used. Only a few arteries underwent patch closure. All operations were performed under general anesthesia and with somatosensory-evoked potential monitoring of cerebral function. Barbiturates were administered during internal carotid artery cross clamping. Patients underwent preoperative DWI imaging ≤24 hours of surgery, with the second study obtained in 92% of patients ≤24 hours postoperatively and all follow up DWI imaging studies obtained ≤72 hours of the operation. MR DWI lesions were assessed by a neuroradiologist. No clinical ischemic complications occurred after CEA and no new DWI ischemic lesions were detected.

Comment: There are, of course, a number of limitations to interpretation of the data here. First of all, it is highly unlikely many vascular surgeons use the technique for CEA described in the article. Two-thirds of the patients were asymptomatic and therefore were likely at low risk for any postoperative ischemic complication. Nevertheless, the series demonstrates it is possible to perform CEA with a rate of new DWI-detected lesions after the procedure that is unlikely to be matched anytime in the near future by carotid artery stenting.

Vascular Function and Circulating Progenitor Cells in Thromboangiitis Obliterans (Buerger's Disease) and Atherosclerosis Obliterans

Idei N, Nishioka K, Soga J. Hypertension 2011;57:70-8.

Conclusion: The number and function of circulating progenitor cells (CPCs) are decreased in patients with atherosclerosis but not in patients with Buerger's disease.

Summary: A healthy endothelium, mediated mainly by nitric oxide, maintains vascular structure and tone by regulating the balance between vasoconstriction and vasodilation as well as growth promotion and growth inhibition and antioxidation and pro-oxidation (Vanhoutte P, Hypertension 1989;13:658-67). Buerger's disease and atherosclerosis are both associated with endothelial dysfunction (Makita S, Circulation 1996;94:11211-5). However, although the mortality rate of patients with atherosclerosis is higher than in age-matched controls, the mortality rate of patients with Buerger's disease is not (Criqui MH, N Engl J Med 1992;326:381-6). The number of CPCs appears to correlate with endothelial function, and the number of CPCs may be a predictor of cardiovascular events (Werner N, N Engl J Med 2005;353:999-1007). Given the discrepancy between apparent cardiovascular death rates in patients with atherosclerosis vs those with Buerger's disease, the authors evaluated CPCs and endothelial function in patients with Buerger's disease and compared them with patients with atherosclerosis. They measured flow-mediated vasodilation (FMD), nitroglycerin-induced vasodilation, and circulating CPCs in 30 patients with Buerger's disease, 30 age- and sex-matched healthy subjects, and in 40 patients with atherosclerosis. In patients with Buerger's disease and in those with atherosclerosis, FMD was less than in the control group (6.6% ± 2.7%, 5.7% ± 3.3% vs 9.5% ± 3.1%, $P < .0001$, respectively). FMD did not differ between patients with Buerger's disease and those with atherosclerosis. Nitroglycerine-induced vasodilation was also similar in the three patient groups. The number and migration of circulating CPCs were similar in the control and Buerger's disease groups but lower in the atherosclerosis group (553 ± 297/mL; 36 ± 18 per high-power field [HPF]) than in those with Buerger's disease (963 ± 543/ml; 62 ± 23 per HPF) and the controls (1063 ± 426/mL, 68 ± 18 per HPF; $P < .0001$). There was a significant relationship between the number and migration of CPCs and FMD ($r = 0.43$ and $r = 0.40$, $P < .0001$, respectively).

Comment: Inflammation is clearly associated with adverse cardiovascular outcomes. Preservation of CPCs in Buerger's disease may allow mitigation of the affects of inflammatory induced oxidative stress and oxidative dysfunction associated with Buerger's disease. In fact, patients with Buerger's disease have a normal number and function of CPCs that may contribute to restoration of endothelial function in these patients, thereby resulting in either reduction or inhibition of the adverse cardiovascular outcomes predicted by the endothelial dysfunction in patients with Buerger's disease.